Trying 3106016892...Open

Welcome to STN International! Enter x:x
LOGINID:ssspta1614jmk
PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2	Sep 29	The Philippines Inventory of Chemicals and Chemical Substances (PICCS) has been added to CHEMLIST
NEWS	3	Oct 27	New Extraction Code PAX now available in Derwent Files
NEWS	4	Oct 27	SET ABBREVIATIONS and SET PLURALS extended in Derwent World Patents Index files
NEWS	5	Oct 27	Patent Assignee Code Dictionary now available in Derwent Patent Files
NEWS	6	Oct 27	Plasdoc Key Serials Dictionary and Echoing added to Derwent Subscriber Files WPIDS and WPIX
NEWS	7	Nov 29	Derwent announces further increase in updates for DWPI

NEWS EXPRESS	FREE UPGRADE 5.0DP1 FOR STN EXPRESS 5.0 WITH DISCOVER! (WINDOWS) NOW AVAILABLE
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS INTER	General Internet Information
NEWS LOGIN	Welcome Banner and News Items
NEWS PHONE	Direct Dial and Telecommunication Network Access to STN
NEWS WWW	CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

* * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 16:28:08 ON 30 NOV 2000

=> file registry

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.15	0.15

FILE 'REGISTRY' ENTERED AT 16:28:26 ON 30 NOV 2000
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

STRUCTURE FILE UPDATES: 29 NOV 2000 HIGHEST RN 305321-79-3
DICTIONARY FILE UPDATES: 29 NOV 2000 HIGHEST RN 305321-79-3

TSCA INFORMATION NOW CURRENT THROUGH July 8, 2000

Please note that search-term pricing does apply when
conducting SmartSELECT searches.

Structure search limits have been increased. See HELP SLIMIT
for details.

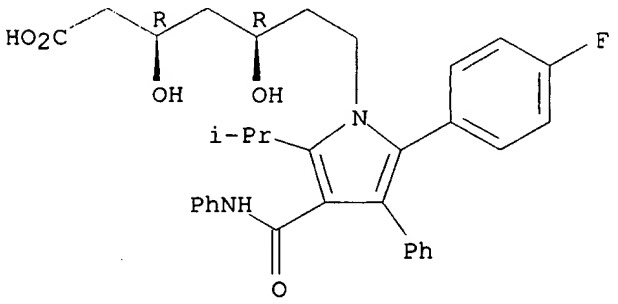
=> s atorvastatin/cn

L1 1 ATORVASTATIN/CN

=> d l1

L1 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2000 ACS
RN 134523-00-5 REGISTRY
CN 1H-Pyrrole-1-heptanoic acid,
2-(4-fluorophenyl)-.beta.,.delta.-dihydroxy-5-
(1-methylethyl)-3-phenyl-4-[(phenylamino)carbonyl]-, (.beta.R,.delta.R)-
(9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 1H-Pyrrole-1-heptanoic acid,
2-(4-fluorophenyl)-.beta.,.delta.-dihydroxy-5-
(1-methylethyl)-3-phenyl-4-[(phenylamino)carbonyl]-, [R-(R*,R*)]-
OTHER NAMES:
CN
(.beta.R,.delta.R)-2-(p-Fluorophenyl)-.beta.,.delta.-dihydroxy-5-isopropyl-
3-phenyl-4-(phenylcarbamoyl)pyrrole-1-heptanoic acid
CN **Atorvastatin**
FS STEREOSEARCH
MF C33 H35 F N2 O5
CI COM
SR CA
LC STN Files: ADISINSIGHT, AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS,
BIOTECHNO, CA, CAPLUS, CBNB, CEN, CIN, DDFU, DIOGENES, DRUGNL, DRUGPAT,
DRUGU, DRUGUPDATES, EMBASE, IMSDIRECTORY, IPA, MRCK*, PROMT, TOXLINE,
TOXLIT, USAN, USPATFULL
(*File contains numerically searchable property data)
Other Sources: WHO

Absolute stereochemistry.



245 REFERENCES IN FILE CA (1967 TO DATE)
9 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
248 REFERENCES IN FILE CAPLUS (1967 TO DATE)

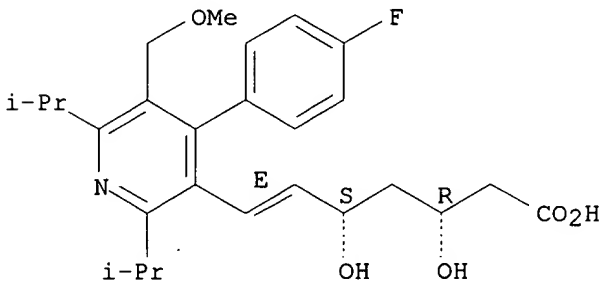
=> s cervastatin/cn

L2 0 CERVASTATIN/CN

=> s cerivastatin/cn

ANSWER 1 OF 1 REGISTRY COPYRIGHT 2003 ACS
RN 145599-86-6 REGISTRY
CN 6-Heptenoic acid, 7-[4-(4-fluorophenyl)-5-(methoxymethyl)-2,6-bis(1-methylethyl)-3-pyridinyl]-3,5-dihydroxy-, (3R,5S,6E)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 6-Heptenoic acid, 7-[4-(4-fluorophenyl)-5-(methoxymethyl)-2,6-bis(1-methylethyl)-3-pyridinyl]-3,5-dihydroxy-, [S-[R*,S*-(E)]]-
OTHER NAMES:
CN (3R,5S,6E)-7-[4-(p-Fluorophenyl)-2,6-diisopropyl-5-(methoxymethyl)-3-pyridyl]-3,5-dihydroxy-6-heptenoic acid
CN **Cerivastatin**
FS STEREOSEARCH
MF C26 H34 F N O5
CI COM
SR World Health Organization
LC STN Files: ADISINSIGHT, ADISNEWS, ANABSTR, BIOBUSINESS, BIOSIS, CA, CAPLUS, CBNB, CEN, CHEMCATS, CIN, CSNB, DDFU, DIOGENES, DRUGNL, DRUGPAT, DRUGU, DRUGUPDATES, IPA, MRCK*, PROMT, SYNTHLINE, TOXCENTER, USAN, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: WHO

Absolute stereochemistry. Rotation (+).
Double bond geometry as shown.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

360 REFERENCES IN FILE CA (1957 TO DATE)
11 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
364 REFERENCES IN FILE CAPLUS (1957 TO DATE)

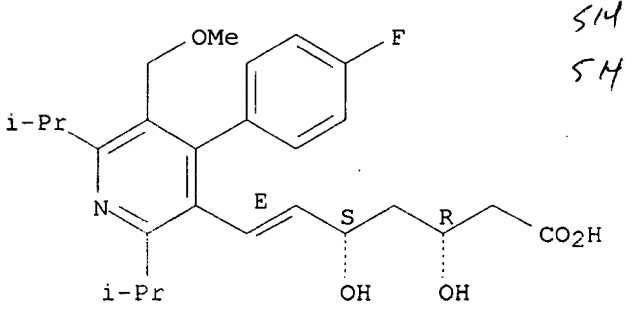
=>

L3 1 CERIVASTATIN/CN

=> d l3

L3 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2000 ACS
RN 145599-86-6 REGISTRY
CN 6-Heptenoic acid, 7-[4-(4-fluorophenyl)-5-(methoxymethyl)-2,6-bis(1-methylethyl)-3-pyridinyl]-3,5-dihydroxy-, (3R,5S,6E)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 6-Heptenoic acid, 7-[4-(4-fluorophenyl)-5-(methoxymethyl)-2,6-bis(1-methylethyl)-3-pyridinyl]-3,5-dihydroxy-, [S-[R*,S*-(E)]]-
OTHER NAMES:
CN (3R,5S,6E)-7-[4-(p-Fluorophenyl)-2,6-diisopropyl-5-(methoxymethyl)-3-pyridyl]-3,5-dihydroxy-6-heptenoic acid
CN **Cerivastatin**
FS STEREOSEARCH
MF C26 H34 F N O5
CI COM
SR World Health Organization
LC STN Files: ADISINSIGHT, BIOBUSINESS, BIOSIS, CA, CAPLUS, CBNB, CEN, CHEMCATS, CIN, DDFU, DRUGNL, DRUGPAT, DRUGU, DRUGUPDATES, IPA, MRCK*, PROMT, TOXLINE, TOXLIT, USAN, USPATFULL
(*File contains numerically searchable property data)
Other Sources: WHO

Absolute stereochemistry. Rotation (+).
Double bond geometry as shown.



514 / 315
514 / 317

119 REFERENCES IN FILE CA (1967 TO DATE)
5 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
119 REFERENCES IN FILE CAPLUS (1967 TO DATE)

=> s l-arginine/cn

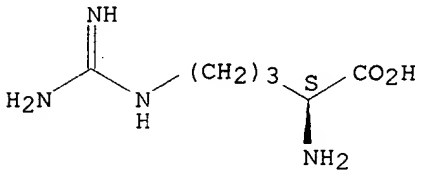
L4 1 L-ARGININE/CN

=> d l4

L4 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2000 ACS
RN 74-79-3 REGISTRY
CN **L-Arginine (9CI)** (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Arginine, L- (8CI)
OTHER NAMES:
CN (S)-2-Amino-5-[(aminoiminomethyl)amino]pentanoic acid
CN 20: PN: US6074850 SEQID: 10 unclaimed sequence
CN 62: PN: WO0000636 PAGE: 70 claimed sequence

CN Arginine
CN L-(+)-Arginine
CN L-.alpha.-Amino-.delta.-guanidinovaleric acid
CN L-Norvaline, 5-[(aminoiminomethyl)amino]-
CN L-Ornithine, N5-(aminoiminomethyl)-
CN Pentanoic acid, 2-amino-5-[(aminoiminomethyl)amino]-, (S)-
FS STEREOSEARCH
DR 7004-12-8, 142-49-4
MF C6 H14 N4 O2
CI COM
LC STN Files: AGRICOLA, AIDSLINE, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS,
BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, DDFU, DETHERM*, DIOGENES,
DRUGU, EMBASE, GMELIN*, HODOC*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PHAR, PIRA, PROMT, RTECS*, SPECINFO, TOXLINE, TOXLIT, TULSA, USAN, USPATFULL, VETU
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



27246 REFERENCES IN FILE CA (1967 TO DATE)
795 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
27299 REFERENCES IN FILE CAPLUS (1967 TO DATE)
6 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

09/419,519

L16 ANSWER 2 OF 104 CAPLUS COPYRIGHT 2001 ACS

AB A review, with 22 refs. The **L-arginine**:nitric oxide pathway is a ubiquitous, second messenger system involved in the regulation of many physiol. processes in animals and humans. Within the cardiovascular system, endothelium-derived NO regulates vascular tone and blood pressure, and modulates the interactions between circulating cells and the blood vessel wall. **Inhibition** of NO synthesis increases vascular tone and blood pressure, and accelerates atherogenesis. In human cardiovascular diseases, abnormalities of the **L-arginine**:NO pathway have been documented that could contribute to the pathophysiol. of diseases such as hypertension, diabetes mellitus, septic shock, and **hypercholesterolemia**. The elucidation of the role of NO in the human cardiovascular system in health and disease could have implications for the development of new therapies.

AN 1997:46474 CAPLUS

DN 126:87361

TI The L-arginine:nitric oxide pathway in the human cardiovascular system

AU MacAllister, Raymond J.; Vallance, Patrick

CS Center Clinical Pharmacology, Univ. College, London Medical Sch., London, UK

SO J. Int. Fed. Clin. Chem. (**1996**), 8(4), 152-158
CODEN: JIFCEM; ISSN: 1051-2292

PB Globetech Publishing

DT Journal; General Review

LA English

TI The L-arginine:nitric oxide pathway in the human cardiovascular system

SO J. Int. Fed. Clin. Chem. (**1996**), 8(4), 152-158
CODEN: JIFCEM; ISSN: 1051-2292

AB A review, with 22 refs. The **L-arginine**:nitric oxide pathway is a ubiquitous, second messenger system involved in the regulation of many physiol. processes in animals and humans. Within the cardiovascular system, endothelium-derived NO regulates vascular tone and blood pressure, and modulates the interactions between circulating cells and the blood vessel wall. **Inhibition** of NO synthesis increases vascular tone and blood pressure, and accelerates atherogenesis. In human cardiovascular diseases, abnormalities of the **L-arginine**:NO pathway have been documented that could contribute to the pathophysiol. of diseases such as hypertension, diabetes mellitus, septic shock, and **hypercholesterolemia**. The elucidation of the role of NO in the human cardiovascular system in health and disease could have implications for the development of new therapies.

L19 ANSWER 37 OF 72 CAPLUS COPYRIGHT 2001 ACS

AB Nitric oxide (NO) may protect arteries against **atherosclerosis**. In the present study, we examd. whether dietary **L-arginine**, the precursor of NO, could chronically preserve endothelium-dependent vasodilation in vivo and/or limit atherogenesis. Rabbits were randomized according to sex to receive 2% dietary **cholesterol**, with or without **L-arginine** (2.25% soln.), for 7 or 14 wk. Hindlimb vasodilator responses to acetylcholine and nitroprusside were measured with an electromagnetic flow probe. **Atherosclerosis** was measured with planimetry of aortic lesions stained with Oil-Red-O. In rabbits administered **L-arginine**, plasma arginine levels increased to 483.+-.30 .mu.mol/L at 3 wk (mean.+-.SEM, P<.0001 vs. control animals) but declined to 224.+-.25 .mu.mol/L at 7 wk (P=.02) and to 100.+-.23 .mu.mol/L at 14 wk (NS vs. control animals). At 7 wk, peak hindlimb conductance in response to acetylcholine in **cholesterol**-fed males was 249.+-.49% of baseline compared with 332.+-.9% in control animals (P=.04), but peak response in arginine-fed rabbits (314.+-.24%) did not differ from that of control animals. At 14 wk, peak responses to acetylcholine were equally **reduced** in males fed **cholesterol** with (266.+-.21%, P=.02 vs. control) or without (263.+-.13%, P=.01 vs. control) **L-arginine**. Similar impairment of endothelium-dependent vasodilatation was seen in females at 14 wk. Vasodilator responses to nitroprusside did not differ from those of control animals in any treatment group. After 14 wk, **atherosclerosis** was less in the descending aorta of arginine-fed males (16.+-.4% surface area) than that of males fed **cholesterol** only (42.+-.8%, P=.04), but no treatment benefit was seen in the ascending aorta or in females. Dietary **L-arginine** supplementation causes an early rise in plasma arginine levels, with limitation of **atherosclerosis** in the descending aorta and preservation of endothelium-dependent vasodilatation in resistance arteries, but this treatment effect is not sustained. Dietary **L-arginine** may not be of longterm benefit in the prevention of **atherosclerosis** in humans.

AN 1996:519828 CAPLUS

DN 125:194272

TI Effects of dietary **L-arginine** on **atherosclerosis** and endothelium-dependent vasodilatation in the **hypercholesterolemic** rabbit: Response according to treatment duration, anatomic site, and sex

AU Jeremy, Richmond W.; McCarron, Hugh; Sullivan, David

CS Department Medicine, University Sydney, Sydney, 2006, Australia

SO Circulation (**1996**), 94(3), 498-506
CODEN: CIRCAZ; ISSN: 0009-7322

DT Journal

LA English

TI Effects of dietary **L-arginine** on **atherosclerosis** and endothelium-dependent vasodilatation in the **hypercholesterolemic** rabbit: Response according to treatment duration, anatomic site, and sex

TI Effects of dietary **L-arginine** on **atherosclerosis** and endothelium-dependent vasodilatation in the **hypercholesterolemic** rabbit: Response according to treatment duration, anatomic site, and sex

SO Circulation (**1996**), 94(3), 498-506
CODEN: CIRCAZ; ISSN: 0009-7322

AB Nitric oxide (NO) may protect arteries against **atherosclerosis**. In the present study, we examd. whether dietary **L-arginine**, the precursor of NO, could chronically preserve

endothelium-dependent vasodilation in vivo and/or limit atherogenesis. Rabbits were randomized according to sex to receive 2% dietary **cholesterol**, with or without **L-arginine** (2.25% soln.), for 7 or 14 wk. Hindlimb vasodilator responses to acetylcholine and nitroprusside were measured with an electromagnetic flow probe. **Atherosclerosis** was measured with planimetry of aortic lesions stained with Oil-Red-O. In rabbits administered **L-arginine**, plasma arginine levels increased to 483.+-.30 .mu.mol/L at 3 wk (mean.+-.SEM, P<.0001 vs. control animals) but declined to 224.+-.25 .mu.mol/L at 7 wk (P=.02) and to 100.+-.23 .mu.mol/L at 14 wk (NS vs. control animals). At 7 wk, peak hindlimb conductance in response to acetylcholine in **cholesterol**-fed males was 249.+-.49% of baseline compared with 332.+-.9% in control animals (P=.04), but peak response in arginine-fed rabbits (314.+-.24%) did not differ from that of control animals. At 14 wk, peak responses to acetylcholine were equally **reduced** in males fed **cholesterol** with (266.+-.21%, P=.02 vs. control) or without (263.+-.13%, P=.01 vs. control) **L-arginine**. Similar impairment of endothelium-dependent vasodilatation was seen in females at 14 wk. Vasodilator responses to nitroprusside did not differ from those of control animals in any treatment group. After 14 wk, **atherosclerosis** was less in the descending aorta of arginine-fed males (16.+-.4% surface area) than that of males fed **cholesterol** only (42.+-.8%, P=.04), but no treatment benefit was seen in the ascending aorta or in females. Dietary **L-arginine** supplementation causes an early rise in plasma arginine levels, with limitation of **atherosclerosis** in the descending aorta and preservation of endothelium-dependent vasodilatation in resistance arteries, but this treatment effect is not sustained. Dietary **L-arginine** may not be of longterm benefit in the prevention of **atherosclerosis** in humans.

IT Sex
(dietary **L-arginine** effect on **atherosclerosis** and endothelium-dependent vasodilatation in the **hypercholesterolemic** rabbit)

IT Arteriosclerosis
(**atherosclerosis**, dietary **L-arginine** effect on **atherosclerosis** and endothelium-dependent vasodilatation in the **hypercholesterolemic** rabbit)

IT Artery
(endothelium, dietary **L-arginine** effect on **atherosclerosis** and endothelium-dependent vasodilatation in the **hypercholesterolemic** rabbit)

IT 74-79-3, Arginine, biological studies
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(dietary **L-arginine** effect on **atherosclerosis** and endothelium-dependent vasodilatation in the **hypercholesterolemic** rabbit)

IT 57-88-5, **Cholesterol**, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(dietary **L-arginine** effect on **atherosclerosis** and endothelium-dependent vasodilatation in the **hypercholesterolemic** rabbit)

=> d l19 39 ab,bib,ti,kwic

L19 ANSWER 39 OF 72 CAPLUS COPYRIGHT 2001 ACS

AB 20 Male NZ white rabbits were enrolled in the 10 wks. randomized controlled expt. to exam. the anti-atherogenic mechanism of dietary **L-arginine**. Plasma **cholesterol** levels of the rabbits fed with high **cholesterol** diet increased 10 folds despite supplementation of **L-arginine** or methionine, and some thickening of the pulmonary artery intima with macrophage infiltration and smooth muscle cell proliferation. Dietary **L-**

arginine supplementation significantly attenuated the thickening of intima, accumulation of macrophages, and proliferation of smooth muscle cells; but methionine did not produce such an effect. The results suggest that dietary **L-arginine** supplementation exerts its anti-atherogenic effect mainly via through **inhibition** of macrophage infiltration and smooth muscle cell proliferation.

AN 1996:588956 CAPLUS
DN 125:274466
TI Dietary **L-arginine** attenuates macrophage infiltration and smooth muscle cell proliferation in the pulmonary artery of **hypercholesterolemic** rabbits
AU Wang, Bingyin; Xu, Chengbin; Cooke, J. P.
CS Dep. of Cardiology, Beijing Medical Univ., Beijing, 100044, Peop. Rep. China
SO Beijing Yike Daxue Xuebao (**1996**), 28(3), 205-207
CODEN: BYDXEV; ISSN: 1000-1530
DT Journal
LA Chinese
TI Dietary **L-arginine** attenuates macrophage infiltration and smooth muscle cell proliferation in the pulmonary artery of **hypercholesterolemic** rabbits
TI Dietary **L-arginine** attenuates macrophage infiltration and smooth muscle cell proliferation in the pulmonary artery of **hypercholesterolemic** rabbits
SO Beijing Yike Daxue Xuebao (**1996**), 28(3), 205-207
CODEN: BYDXEV; ISSN: 1000-1530
AB 20 Male NZ white rabbits were enrolled in the 10 wks. randomized controlled expt. to exam. the anti-atherogenic mechanism of dietary **L-arginine**. Plasma **cholesterol** levels of the rabbits fed with high **cholesterol** diet increased 10 folds despite supplementation of **L-arginine** or methionine, and some thickening of the pulmonary artery intima with macrophage infiltration and smooth muscle cell proliferation. Dietary **L-arginine** supplementation significantly attenuated the thickening of intima, accumulation of macrophages, and proliferation of smooth muscle cells; but methionine did not produce such an effect. The results suggest that dietary **L-arginine** supplementation exerts its anti-atherogenic effect mainly via through **inhibition** of macrophage infiltration and smooth muscle cell proliferation.
IT Cell proliferation
Macrophage
(dietary L-arginine supplementation has an anti-atherogenic effect by **inhibiting** macrophage infiltration and smooth muscle cell proliferation)
IT Antiarteriosclerotics
(antiatherosclerotics, dietary L-arginine supplementation has an anti-atherogenic effect by **inhibiting** macrophage infiltration and smooth muscle cell proliferation)
IT Arteriosclerosis
(**atherosclerosis**, dietary **L-arginine** supplementation has an anti-atherogenic effect by **inhibiting** macrophage infiltration and smooth muscle cell proliferation)
IT Artery
(pulmonary, intima; dietary L-arginine supplementation has an anti-atherogenic effect by **inhibiting** macrophage infiltration and smooth muscle cell proliferation)
IT 74-79-3, L-Arginine, biological studies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(dietary L-arginine supplementation has an anti-atherogenic effect by **inhibiting** macrophage infiltration and smooth muscle cell proliferation)
IT 57-88-5, **Cholesterol**, biological studies

RL: ADV (Adverse effect, including toxicity); BOC (Biological occurrence);
BIOL (Biological study); OCCU (Occurrence)
(metabolic disorders, **hypercholesterolemia**; dietary **L-arginine** supplementation has an anti-atherogenic effect by **inhibiting** macrophage infiltration and smooth muscle cell proliferation)

=> d l19 57 ab,bib,ti,kwic

L19 ANSWER 57 OF 72 CAPLUS COPYRIGHT 2001 ACS
AB Dietary supplementation with 2.25% **L-arginine** in the drinking water had antiatherogenic effects in **hypercholesterolemic** rabbits without affecting serum **cholesterol** levels.
AN 1995:324112 CAPLUS
DN 122:96212
TI Antiatherogenic effects of **L-arginine** in **hypercholesterolemic** rabbits
AU Wang, Bingyin; Xu, Chengkin; Cooke, P. John; Kosak, Jon
CS People's Hospital, Beijing Med. Univ., Beijing, Peop. Rep. China
SO Beijing Yike Daxue Xuebao (**1994**), 26(3), 189-91
CODEN: BYDXEV; ISSN: 1000-1530
DT Journal
LA Chinese
TI Antiatherogenic effects of **L-arginine** in **hypercholesterolemic** rabbits
TI Antiatherogenic effects of **L-arginine** in **hypercholesterolemic** rabbits
SO Beijing Yike Daxue Xuebao (**1994**), 26(3), 189-91
CODEN: BYDXEV; ISSN: 1000-1530
AB Dietary supplementation with 2.25% **L-arginine** in the drinking water had antiatherogenic effects in **hypercholesterolemic** rabbits without affecting serum **cholesterol** levels.
ST atherosclerosis **inhibition** hypercholesterolemia arginine
IT Antiarteriosclerotics
(atherosclerosis **inhibition** by arginine in hypercholesterolemia)
IT 74-79-3, **L-Arginine**, biological studies
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**atherosclerosis inhibition** by arginine in **hypercholesterolemia**)
IT 57-88-5, Cholesterol, biological studies
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(hypercholesterolemia; atherosclerosis **inhibition** by arginine in hypercholesterolemia)

=> d l19 60 ab,bib,ti,kwic

L19 ANSWER 60 OF 72 CAPLUS COPYRIGHT 2001 ACS
AB **Hypercholesterolemia**, before **atherosclerosis**, is known to **reduce** agonist- (e.g., acetylcholine) mediated nitric oxide (NO) prodn. within 2 wk of a **cholesterol**-enriched diet. However, no data exist on the effect of **hypercholesterolemia** on the basal release of NO from blood vessels. The authors studied the basal release of NO in rabbit coronary arteries by addn. of the NO synthase blocker NG-nitro-**L-arginine**-Me ester (L-NAME). Basal release of NO was markedly attenuated 2 wk after introduction of a 0.5%

cholesterol addn. to the diet. One week later, the adherence of neutrophils to the coronary endothelium was enhanced. The increased adhesiveness could be attributed to enhanced endothelial adhesion rather than to changes in the properties of the leukocytes. Both phenomena could be reversed by addn. of **L-arginine** to isolated coronary arteries. Administration of 10 mg/day lovastatin, a 3-hydroxy-3-methylglutaryl CoA reductase **inhibitor**, markedly attenuated both the **reduced** basal NO prodn. and the increased adhesiveness of the endothelium. These results support the concept that NO is an important protective agent produced by the endothelium to preserve the integrity of the endothelium and may protect it against atherogenesis.

AN 1993:623424 CAPLUS
DN 119:223424
TI **Decreased** basal nitric oxide release in **hypercholesterolemia** increases neutrophil adherence to rabbit coronary artery endothelium
AU Lefer, Allan M.; Ma, Xin Liang
CS Jefferson Med. Coll., Thomas Jefferson Univ., Philadelphia, PA, 19107-6799, USA
SO Arterioscler. Thromb. (**1993**), 13(6), 771-6
CODEN: ARTTE5; ISSN: 1049-8834
DT Journal
LA English
TI **Decreased** basal nitric oxide release in **hypercholesterolemia** increases neutrophil adherence to rabbit coronary artery endothelium
TI **Decreased** basal nitric oxide release in **hypercholesterolemia** increases neutrophil adherence to rabbit coronary artery endothelium
SO Arterioscler. Thromb. (**1993**), 13(6), 771-6
CODEN: ARTTE5; ISSN: 1049-8834
AB **Hypercholesterolemia**, before **atherosclerosis**, is known to **reduce** agonist- (e.g., acetylcholine) mediated nitric oxide (NO) prodn. within 2 wk of a **cholesterol**-enriched diet. However, no data exist on the effect of **hypercholesterolemia** on the basal release of NO from blood vessels. The authors studied the basal release of NO in rabbit coronary arteries by addn. of the NO synthase blocker NG-nitro-**L-arginine**-Me ester (L-NAME). Basal release of NO was markedly attenuated 2 wk after introduction of a 0.5% **cholesterol** addn. to the diet. One week later, the adherence of neutrophils to the coronary endothelium was enhanced. The increased adhesiveness could be attributed to enhanced endothelial adhesion rather than to changes in the properties of the leukocytes. Both phenomena could be reversed by addn. of **L-arginine** to isolated coronary arteries. Administration of 10 mg/day lovastatin, a 3-hydroxy-3-methylglutaryl CoA reductase **inhibitor**, markedly attenuated both the **reduced** basal NO prodn. and the increased adhesiveness of the endothelium. These results support the concept that NO is an important protective agent produced by the endothelium to preserve the integrity of the endothelium and may protect it against atherogenesis.

cardiac insufficiency after myocardial infarction, diabetic nephropathy,. . . vascular hypertrophy or obstruction after percutaneous transluminal coronary angioplasty, vascular reobstruction after bypass surgery, hyperaldosteronism, glomerulosclerosis, renal insufficiency, glaucoma, occular **hypertension**, hyperlipemia, myocardial infarction, angina pectoris, aneurysm, coronary arteriosclerosis, cerebral arteriosclerosis, peripheral arteriosclerosis, thrombosis, diseases of central nervous system, Alzheimer's disease,. . .

. . . The method as claimed in claim 1, wherein said method is directed to the prevention or treatment of complications of **hypertension**.

10. A method as claimed in claim 1, wherein said method is for the prevention or treatment of **hypertension**, arteriosclerosis or hyperlipemia and which said method comprises administering an effective amount of (.+-.)-1-(cyclohexyloxycarbonyloxy)ethyl 2-ethoxy-1-[[2'-(1H-tetrazol-5-yl)biphenyl-4-yl]methyl]-1H-benzimidazole-7-carboxylate or a salt. . .

11. A method as claimed in claim 1, wherein said method is for the prevention or treatment of **hypertension**, arteriosclerosis or hyperlipemia and which said method comprises administering an effective amount of 2-ethoxy-1-[[2'-(1H-tetrazol-5-yl)biphenyl-4-yl]methyl]-1H-benzimidazole-7-carboxylic acid or a salt thereof. . .

12. A method as claimed in claim 1, wherein said method is for the prevention or treatment of **hypertension**, arteriosclerosis or hyperlipemia and which said method comprises administering an effective amount of 2-ethoxy-1-[[2'-(2,5-dihydro-5-oxo- 1,2 ,4-oxadiazol-3-yl)biphenyl-4-yl]methyl]-1H-benzimidazole-7-carboxylic acid or a salt. . .

IT 83435-66-9 83480-29-9 111025-46-8 139481-59-7 145040-37-5
145599-86-6 147403-03-0 178610-08-7
(pharmaceutical compns. contg. angiotensin II antagonists and addnl. agents for treatment of angiotensin II-mediated diseases)

=>

ANSWER 1 OF 1 USPATFULL
AN 2000:109828 USPATFULL
TI Pharmaceutical composition
IN Tamura, Norikazu, Kobe, Japan
Sohda, Takashi, Takatsuki, Japan
Ikeda, Hitoshi, Higashiosaka, Japan
PA Takeda Chemical Industries, Ltd., Osaka, Japan (non-U.S. corporation)
PI US 6107323 20000822
WO 9737688 19971016 <--
AI US 1997-836784 19970516 (8)
WO 1997-JP1149 19970403
19970516 PCT 371 date
19970516 PCT 102(e) date
PRAI JP 1996-83917 19960405
DT Utility
FS Granted
EXNAM Primary Examiner: Travers, Russell
LREP Foley & Lardner
CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1625
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB To provide a pharmaceutical composition which performs a remarkable effect with a relatively decreased dosage, and, with less side effects, a pharmaceutical composition formulated by combination of an angiotensin II-mediated compound or a salt thereof with at least one species of a compound having the activity of increasing insulin-sensitivity, a compound having the activity of improving postprandial hyperglycemia in diabetes mellitus, an indane derivative having the activity of inhibiting angiotensin converting enzyme, a pyridine derivative having the activity of inhibiting HMG-Co A reductase or salts thereof are advantageously employed.
PI US 6107323 20000822
WO 9737688 19971016 <--
SUMM . . . antagonizes to this angiotensin II at angiotensin II receptor, is useful for the prophylaxis and therapy of circulatory diseases including **hypertension**, cardiac diseases (e.g. heart failure, myocardial infarction, etc.), cerebral apoplexy, nephritis, arteriosclerosis, etc. And, an angiotensin converting enzyme drug suppresses. . . II, which is considered, like angiotensin II antagonistic drugs, as useful for the prophylaxis and therapy of circulatory diseases including **hypertension**, cardiac diseases (e.g. heart failure, myocardial infarction, etc.), cerebral apoplexy, nephritis, arteriosclerosis, etc. However, since angiotensin converting enzyme is the. . .
SUMM Above all, such diseases as **hypertension**, abnormal carbohydrate tolerance and abnormal lipid metabolism have been known to be complicated with one another. Especially, **hypertension** and insulin resistance, or **hypertension** and arteriosclerosis are considered to aggravate the respective counterpart diseases.
SUMM . . . with a compound having action mechanism other than the above, to perform especially remarkable effects in angiotensin II-mediated diseases, especially **hypertension**, hyperlipemia, arteriosclerosis and so on, singly or complications of these diseases and to cover up various defects observed in administration. . .
SUMM (4) the composition as described in the above (2), which is directed to the prophylaxis (prevention) or therapy (treatment) of **hypertension**, cardiac insufficiency, cerebral apoplexy, ischemic peripheral circulation disturbances, myocardial ischemia, venous insufficiency, progressive cardiac insufficiency after myocardial infarction, diabetic nephropathy,. . . vascular hypertrophy or

obstruction after percutaneous transluminal coronary angioplasty, vascular reobstruction after bypass surgery, hyperaldosteronism, glomerulosclerosis, renal insufficiency, glaucoma, occular **hypertension**, hyperlipemia, myocardial infarction, angina pectoris, aneurysm, coronary arteriosclerosis, cerebral arteriosclerosis, peripheral arteriosclerosis, thrombosis, diseases of central nervous system, Alzheimer's disease,. . .

SUMM (5) the composition as described in the above (2), which is directed to the prophylaxis or therapy of complications of **hypertension**;

SUMM (17) a pharmaceutical composition for the prevention or treatment of **hypertension**, arteriosclerosis or hyperlipemia comprising (.+-.)-1-(cyclohexyloxycarbonyloxy)ethyl 2-ethoxy-1-[[2'-(1H-tetrazol-5-yl)biphenyl-4-yl]methyl]-1H-benzimidazole-7-carboxylate or a salt thereof in combination with at least one species selected from the. . .

SUMM (18) a pharmaceutical composition for the prevention or treatment of **hypertension**, arteriosclerosis or hyperlipemia comprising 2-ethoxy-1-[[2'-(1H-tetrazol-5-yl)biphenyl-4-yl]methyl]-1H-benzimidazole-7-carboxylic acid or a salt thereof in combination with at least one species selected from the. . .

SUMM (19) a pharmaceutical composition for the prevention or treatment of **hypertension**, arteriosclerosis or hyperlipemia comprising 2-ethoxy-1-[[2'-(2,5-dihydro-5-oxo-1,2,4-oxadiazol-3-yl)biphenyl-4-yl]methyl]-1H-benzimidazole-7-carboxylic acid or a salt thereof in combination with at least one species selected from the. . .

SUMM . . . methanesulfonic acid, benzenesulfonic acid, p-toluenesulfonic acid, etc. Preferable examples of a salt with a basic amino acid include salts with **arginine**, lysine, ornithine, etc. Preferable examples of a salt with an acidic amino acid include salts with aspartic acid, glutamic acid,. . .

SUMM . . . (+)-3R,5S-erythro-(E)-7-[4-(4-fluorophenyl)-2,6-diisopropyl-5-methoxymethylpyridin-yl]-3,5-dihydroxyhept-6-enoic acid or salts thereof. These preferred combinations (1) to (3) are preferably used for the prevention or treatment of **hypertension**, arteriosclerosis or hyperlipemia, in particular, arteriosclerosis accompanied with **hypertension**.

SUMM . . . angiotensin II-mediated diseases of animals, especially mammals (e.g. man, dog, rabbit, rat, mouse, etc.), as exemplified by circulatory diseases including **hypertension**, cardiac insufficiency, cerebral apoplexy, ischemic peripheral circulation disturbances, myocardial ischemia, venous insufficiency, progressive cardiac insufficiency after myocardial infarction, diabetic nephropathy,. . . vascular hypertrophy or obstruction after percutaneous transluminal coronary angioplasty, vascular reobstruction after bypass surgery, hyperaldosteronism, glomerulosclerosis, renal insufficiency, glaucoma, occular **hypertension**, hyperlipemia, myocardial infarction, angina pectoris, aneurysm, coronary arteriosclerosis, cerebral arteriosclerosis, peripheral arteriosclerosis, thrombosis; diseases of sensory disturbances including Alzheimer's disease,. . .

SUMM . . . of this invention performs remarkable effects for the prophylaxis or therapy of diseases accompanied with diabetic, obesitic, hyperlipemic or essential **hypertension**. It is preferably used, especially, for the prophylaxis or therapy of arteriosclerosis accompanied with **hypertension**.

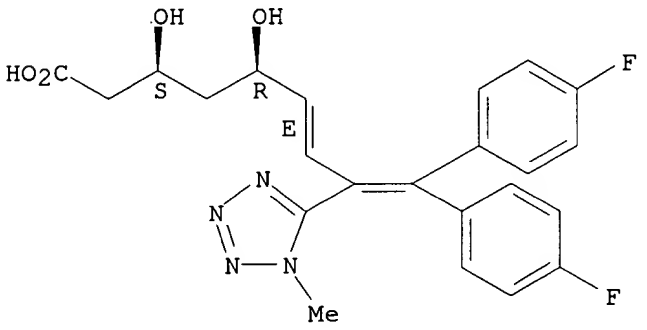
DETD . . . and can be advantageously used as a prophylactic or therapeutic agent of angiotensin II-mediated diseases, especially arteriosclerosis or arteriosclerosis having **hypertension** as a complication.

CLM What is claimed is:

3. The method as claimed in claim 1, wherein said method is directed to the prevention or treatment of **hypertension**, cardiac insufficiency, cerebral apoplexy, ischemic peripheral circulation disturbances, myocardial ischemia, venous insufficiency, progressive

L4 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2003 ACS
RN 129829-03-4 REGISTRY
CN 6,8-Nonadienoic acid, 9,9-bis(4-fluorophenyl)-3,5-dihydroxy-8-(1-methyl-1H-tetrazol-5-yl)-, (3R,5S,6E)-rel- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 6,8-Nonadienoic acid, 9,9-bis(4-fluorophenyl)-3,5-dihydroxy-8-(1-methyl-1H-tetrazol-5-yl)-, [R*,S*-(E)]-(.+-.)-
OTHER NAMES:
CN (.+-.)-BMY-21950
CN 6,8-Nonadienoic acid, 9,9-bis(4-fluorophenyl)-3,5-dihydroxy-8-(1-methyl-1H-tetrazol-5-yl)-, [R*,S*-(E)]-
CN **BMS 180431**
CN BMY 21950
FS STEREOSEARCH
MF C23 H22 F2 N4 O4
CI COM
SR CA
LC STN Files: ADISINSIGHT, BEILSTEIN*, BIOBUSINESS, BIOSIS, CA, CAPLUS, DRUGNL, DRUGUPDATES, IPA, PHAR, PROMT, USPAT2, USPATFULL
(*File contains numerically searchable property data)

Relative stereochemistry.
Double bond geometry as shown.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

9 REFERENCES IN FILE CA (1957 TO DATE)
3 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
9 REFERENCES IN FILE CAPLUS (1957 TO DATE)

=>